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ALCOHOL, DIPHTHERIA, ETC.

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HOSPITAL, TO THE METHODIST EPISCOPAL HOSPITAL, AND
TO THE HOME FOR CRIPPLED CHILDREN.

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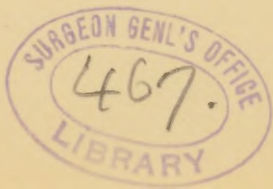
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**FORMS OF PSEUDO-TABES DUE TO LEAD,
ALCOHOL, DIPHTHERIA, ETC.¹**

BY JAMES HENDRIE LLOYD, M.D.,
PHYSICIAN TO THE NERVOUS AND INSANE DEPARTMENT OF THE PHILA-
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LOCOMOTOR ataxia is one of the best-known diseases. Its classic array of symptoms, so familiar to all, occurs, as a rule, with a precision of type and regularity of sequence that admit of but few errors in diagnosis. Its history and course have been written so frequently and so well that little remains to be said; while, unfortunately, its prognosis admits of but such a frank statement of despair, that it has long been described as one of the most inveterate and progressive of diseases. Locomotor ataxia, however, like many other diseases, has its counterfeits. All is not necessarily lost, as was once taught, to all men who stagger in the dark, whose knee-jerks are abolished, and who have pain and numbness in the legs. These counterfeits, doubtless, are in the minority to the true ataxics, but that they are more prevalent than was formerly supposed is at least probable; and that it is highly important for the welfare of the patient and the credit of the physi-

¹ Read before the College of Physicians of Philadelphia, March 2, 1892.



cian to differentiate them, is apparent. This paper will discuss especially the etiology and diagnosis of these forms of pseudo-tabes, and will be based largely upon personal observation.

In 1884 Dejerine¹ described a disease which he called *nervo-tabes peripherique*, and gave the clinical histories and autopsies of two patients whose disease he had diagnosticated in life as locomotor ataxia. Both of these patients presented incoördination, anesthesia, abolition of knee-jerks, slight atrophy, and paresis, without eye or bladder symptoms. They had used alcohol to excess. The autopsies revealed pronounced inflammation of the cutaneous nerve-endings, slighter changes in the intramuscular nerves, and no change whatever in the spinal cord, nerve-roots, or ganglia. Dejerine had not found any analogous cases reported at that time in medical literature, and deserves credit for first demonstrating the striking resemblance of these obscure cases to locomotor ataxia. At about the same time, however, Dreschfeld² described a type of alcoholic ataxia characterized by incoördination in the gait, absence of knee-jerks, with lancinating pains, but without atrophy or paralysis. His clinical picture was strikingly like that of Dejerine, but was not supplemented by the same careful post-mortem findings. In the same year Krüche wrote a paper on "Pseudo-Tabes in Alcoholics."³ But earlier than all of these, Samuel Wilks, in his lectures in 1878, described the drunkard's paraplegia, and said that

¹ Arch. de Physiologie Norm. et Patholog., 3e. sér., iii, 1884.

² Brain, July, 1884, p. 201.

³ Deutsche med. Zeitung, 1884, No. 72, p. 229.

in some cases it closely resembled locomotor ataxia. Wilks believed that the lesion was in the spinal cord, but he wrote before multiple neuritis was much more than dreamed of.

Still earlier (1867) Leudet¹ wrote an elaborate paper on chronic alcoholism in which he described the same combination of symptoms—anesthesia, hyperesthesia, disordered gait, and paresis—and in fact came near to describing what we recognize to-day as polyneuritis. In recent years the literature of multiple neuritis has grown to a vast extent. It has become, therefore, more and more necessary to differentiate the various types of the disease and to recognize its many causes. With this motive, Leyden in 1888 wrote a monograph in which he distinguished five forms, one of which he named the sensory form, under which heading he grouped the acute ataxics, and borrowed for them, from the earlier paper of Dejerine, the title of *neuro-tabes peripherica*.

It may be said in general terms that all of these cases of pseudo-tabes or acute ataxia are instances of a multiple neuritis of this sensory form of Leyden, and that most of them have probably a morbid anatomy similar to or identical with that demonstrated by Dejerine, *i. e.*, an involvement especially of the peripheral distribution of the cutaneous nerves. Hence the most common symptoms in these cases are disorders of sensation, especially anesthesia and paræsthesia, with ataxia and abolition of the knee-jerk. To these must be added symptoms, in varying in-

¹ "Étude clinique de la forme hyperesthésique de l'Alcoolisme chronique, etc.," Arch. Gén. de Méd., 1867, vol. i, pp. 5-35.

tensity, of involvement of the motor nerves, never perhaps quite absent, but often requiring careful and expert examination for their detection. From these ataxic cases all degrees of severity occur up to well-recognized cases of multiple neuritis, with general involvement of the sensory and motor nerves. The eye and bladder are seldom involved in these ataxic cases, but important exceptions occur. Of the great viscera, the heart and kidneys are most likely to suffer.

In some cases it is quite impossible at once to arrive at a correct diagnosis. In doubtful cases the history alone may determine the diagnosis; and this history, with certain attendant phenomena—as, for instance, the blue line on the gums in lead cases, tachycardia in alcoholic cases, and paralysis of accommodation in post-diphtheritic cases—should always be most carefully investigated. In my observation by far the most frequent causes are alcohol, lead, and diphtheria. Others report arsenic, and such infectious processes as variola, typhoid fever, tuberculosis, and syphilis; also wasting diseases, as diarrhea and dysentery. Bartholow has reported cases of paralysis following bowel-disorder, which were probably examples of neuritis. Syphilis is now believed by many to be capable of causing neuritis; indeed, it may be a question whether some cases of so-called syphilitic locomotor ataxia, cured by mercury and iodides, have not been cases of peripheral neuritis. Dejerine, in another paper, described the onset of a fatal multiple neuritis in a morphine-taker; and I have myself seen amblyopia

accompanied by anesthesia of the feet and legs in an excessive smoker.

The eye-symptoms are the most reliable for purposes of differentiation. None of the forms of multiple neuritis, except the post-diphtheritic kind, present, as a rule, any affection of the internal or external ocular muscles. I have looked for them always in vain in alcoholic and lead cases. In diphtheritic paralysis, however, the ciliary muscle is often paralyzed, while the light-reflex remains—the very opposite to the Argyll-Robertson pupil seen in locomotor ataxia. At the same time there is likely to be paralysis of some of the external ocular muscles, causing strabismus; almost always associated with paralysis of the velum palati. In lead-poisoning optic neuritis is sometimes observed.

The functions of the bladder and sexual apparatus are not often affected in any form of polyneuritis, especially the ataxic form. The only case in which I have seen impairment of the bladder was in a woman with grave alcoholic paralysis, with mental symptoms, and weak, rapid heart. The involuntary passage of urine in her case was the result of her mental condition. In chronic inebriates the sexual appetite rather than the sexual power is impaired. On the other hand, locomotor ataxia, as is well known, is very likely to exhibit soon in the case impairment of sexual power and of the expulsive power of the bladder.

It is most probable that ataxia or incoördination, whether in true tabes dorsalis or in pseudo-tabes, is caused by the impairment of sensation. However caused, it is very similar in the two conditions.

We have at present in the Philadelphia Hospital a man suffering with a severe grade of multiple neuritis, with wasting of the muscles of the extremities. He has an alcoholic history, although, strange to say, his disease developed rapidly after a sunstroke. This man has diminished and sluggish, not abolished, knee-jerks. His gait is typically ataxic, with elevation and flapping of the feet. He cannot stand an instant with his eyes closed; in fact, he has such a condition of *astasia* that he is sometimes compelled to keep walking to avoid falling. His eyes are normal. It has been claimed by some that the element of muscular weakness, in cases of neuritis, admits of a distinction being made between the ataxia of the two conditions, as in multiple neuritis the ataxic movement is evidently more feeble than in tabes dorsalis, and the foot is lifted higher because of the paralysis of the extensors. But in the cases of pseudo-tabes, this muscular weakness is often not conspicuous, and hence this distinction is not possible. I have recently put side by side a case of acute lead-poisoning and a typical case of locomotor ataxia for the purpose of comparing the ataxic movements in the arms, which are very marked in both cases.

The cases are as follows:

Male, aged thirty-eight years. After four months' exposure in a lead factory he began to have colic, constipation, vertigo, and headache. He then had several convulsions, and passed into a state of lead encephalopathy, from which he is now recovering. He has no paralysis of the extensors, but the shoulder muscles and biceps are paretic. He has some

patches of anesthesia on the arms, hands, and legs. His walk is ataxic, and the movements of his arms markedly so. He has the blue line on the gums, no albumin or casts in the urine, and the margins of the optic discs are slightly clouded.

This man's case has been a grave one, and admits of no confusion. The ataxic movements of his arms, however, are almost identical with those of the second case, as follows:

Male, aged thirty-eight years. He may be called a case of rapid acute locomotor ataxia. The disease began with numbness and pricking sensations. The patient was soon suffering with fulgurant pains; then ataxia, with inability to walk in the dark. The arms are involved as well as the legs. The knee-jerks are abolished. For a year he has had nocturnal incontinence of urine, and when the bowels are overloaded, some paresis of the sphincter ani. The pupils are unequal and do not respond to light. There are irregular discs, with contracted arteries. There is apparently some latent iritis, and there is a posterior synechia. The man has had syphilis.

The ataxic movements in these two men are so similar as to attract attention, the only difference being that the man with lead-poisoning takes longer to perform a given action than the tabetic, probably because of the element of muscular atrophy in his case.

Motor symptoms, other than ataxia, are not conspicuous in cases simulating tabes; in fact it is the absence of marked paralysis in these cases of multiple neuritis that more than anything causes them to resemble cases of true locomotor ataxia. This was so in Dejerine's cases already referred to, in

which the autopsies showed the principal changes to be in the cutaneous nerves. These are the cases which Leyden classes as his sensory forms, and calls acute ataxics. They are more often alcoholic and diphtheritic cases than any others. It is most probable, however, that none of these cases are entirely exempt from paresis in some muscles. Careful examination usually reveals some loss in muscular masses, and electrical tests sometimes solve a doubt. Paralysis may be slight, especially in the large muscles of the limbs, and may not be conspicuous when the patient is lying down, but when standing up or when executing fine movements with the fingers and toes the motor impairment is more marked.

I recall the case of a man in middle life who had diphtheritic ataxia, with anesthesia, but who had so little paralysis that he was able to be about without attracting attention. Such cases in middle life are especially likely to be diagnosticated as cases of locomotor ataxia.

In diphtheritic cases, acute sensory symptoms, such as pain on pressure over nerve-trunks and on handling the limbs, are not nearly so marked as in alcoholic cases. In all cases, however, presenting true ataxia from whatever cause, I believe a diminution or alteration of sensory function can be demonstrated by careful examination.

Rapidity of evolution is not an absolutely sure sign that the disease is multiple neuritis, because true tabes may evolve rapidly. I have under observation a case of apparently true tabes in a ship-carpenter which began and continued as follows :

Two years since, while crossing the equator, being much overheated, he took a shower-bath, by having buckets of water thrown over him. He was in good health at the time. Three hours later he began to have tingling sensations in the extremities, which he says were at first confined to one side. In a few days he was paralyzed. Later, the other side was affected, but never so badly as the right side. At present he presents a very ataxic gait, pains in the legs and back, and abolition of the knee-jerks. One pupil is twice as large as the other, the dilated iris being immobile, and the other showing the Argyll-Robertson phenomenon. He cannot stand with his eyes shut. His control of his bladder and rectum is impaired, and one ankle is enlarged somewhat like a tabetic joint. The muscular nutrition in the arms and legs is not bad, and his grip in each hand registers 80°. The man has had destructive nose-disease, but denies syphilis.

This patient's history suggests neuritis, but his present state is so exactly that of locomotor ataxia, with eye and bladder symptoms, that the conclusion is almost forced that such is the correct diagnosis.

The case of the patient from whose cord and nerves I present sections, has interest because the diagnosis of locomotor ataxia was actually made by a very expert clinician.

I. L., aged fifty-eight years, was a painter by trade. He had been exposed to lead for thirty years. He had a very clear history of many attacks of colic, constant constipation, and several attacks of wrist-drop. He had also the blue line on the gums, and that frequent result of chronic plumbism, a contracted kidney, as shown by casts, albumin, and the specific gravity of his urine. No lead could be

detected in his urine. He had been an excessive drinker, but had never had syphilis. The diagnosis with which he was sent to me had been made largely upon the ataxic gait, swaying with closed eyes, abolition of the patellar reflexes and numbness, tingling and retarded sensation in the soles. I found his real condition as follows: In addition to the foregoing very evident symptoms, he had well-marked paresis and atrophy of both the extensors and flexors of the forearms and hands, as well as the biceps and deltoid muscles. On voluntary motion there was tremor rather than ataxia of the arms and hands. There were paresthesia and areas of anesthesia on the hands, legs, and soles of the feet. The legs and arms were slightly contracted. The external and internal eye-muscles were normal. Sexual power was normal, and the control of the bladder was perfect. The gait was ataxic, but flapping of the feet was not marked. The hands and feet were slightly edematous. There were partial reaction of degeneration and abolition of faradic contractility in all of the paretic muscles. Optic atrophy was noted by Dr. Gould—no doubt the result of optic neuritis due to lead. The diagnosis was chronic lead-poisoning, with multiple neuritis and contracted kidneys. The patient died in a few weeks in uremic coma. The sections show quite extensive inflammation, or degeneration due to inflammation, in the several nerve-trunks examined, with involvement, rather diffused, of some areas of the cord, notably in the lumbar region. The systemic changes of locomotor ataxia are not present.

In studying this case, in which it seems probable that the cord was involved later than the nerves, I have been reminded of the suggestion made by Leyden, that true *tabes dorsalis* may be secondary

to peripheral neuritis of the sensory or cutaneous form. This is still further demonstrated in a recent study by Pal,¹ who, in a series of cases of multiple neuritis due to alcohol, arsenic, typhoid infection, etc., found apparently secondary changes in the posterior columns and other parts of the cord. It is also well to recall that locomotor ataxia is sometimes complicated by an attack of acute multiple neuritis.

¹ Wiener klin. Wochenschr., 1891. Abstracted in Journal of Nervous and Mental Diseases, March, 1892, p. 225.

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